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 FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 08:57:53 ON 09 JUN 2005
L1            0 S RNR1 AND ALBICAN
L2           85 S ALBICAN
L3        60637 S CANDIDA (2W) ALBICANS
L4           13 S ABLICAN?
L5        63338 S ALBICAN?
L6        26751 S DEBACKER?/AU OR NELISSEN?/AU OR LOGGHE?/AU OR LUYTEN?/AU OR W
L7        24939 S ANTIFUNDAL OR FUNGICIDE
L8        32987 S SCREENING (3W) (METHOD OR PROCESS OR ASSAY)
L9           32 S L7 AND L8
L10           1 S L9 AND YEAST
L11         134 S PROMOTER (3W) INTERFERENCE
L12        2220 S ANTISENSE (3W) INHIBITION
L13           3 S L11 AND L12
L14           1 DUP REM L13 (2 DUPLICATES REMOVED)
L15           3 S L12 AND L3
L16           1 DUP REM L15 (2 DUPLICATES REMOVED)
L17          20 S L9 NOT PY>=2001
L18          18 DUP REM L17 (2 DUPLICATES REMOVED)
L19         108 S RNR1
L20         174 S SAM1 OR SAM2
L21           1 S L20 AND L5
L22           0 S L19 AND L5
L23      174837 S CEREVISIAE
L24          68 S L23 AND L19
L25          38 S L23 AND L20
L26          41 S L24 NOT PY>=2000
L27          26 S L25 NOT PY>=2000
L28          20 DUP REM L25 (18 DUPLICATES REMOVED)
L29          19 DUP REM L26 (22 DUPLICATES REMOVED)
L30          13 DUP REM L27 (13 DUPLICATES REMOVED)
L31      102880 S STRUCTURE? (3W) FUNCTION?
L32           0 S L30 AND L31
L33           0 S L29 AND L31

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L38 ANSWER 2 OF 29 MEDLINE on STN
ACCESSION NUMBER: 1999126418 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9927443
TITLE: Interaction between the MEC1-dependent DNA synthesis checkpoint and G1 cyclin function in Saccharomyces **cerevisiae**.
AUTHOR: Vallen E A; Cross F R
CORPORATE SOURCE: Department of Biology, Swarthmore College, Swarthmore, Pennsylvania 19081, USA.. evallen1@swarthmore.edu
CONTRACT NUMBER: GM47238 (NIGMS)
GM54300-01 (NIGMS)
SOURCE: Genetics, (1999 Feb) 151 (2) 459-71.
Journal code: 0374636. ISSN: 0016-6731.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990324
Last Updated on STN: 20020907
Entered Medline: 19990309

AB The completion of DNA synthesis in yeast is monitored by a checkpoint that requires MEC1 and RAD53. Here we show that deletion of the Saccharomyces **cerevisiae** G1 cyclins CLN1 and CLN2 suppressed the essential requirement for MEC1 function. Wild-type levels of CLN1 and CLN2, or overexpression of CLN1, CLN2, or CLB5, but not CLN3, killed mec1 strains. We identified **RNR1**, which encodes a subunit of ribonucleotide reductase, as a high-copy suppressor of the lethality of mec1 GAL1-CLN1. Northern analysis demonstrated that **RNR1** expression is reduced by CLN1 or CLN2 overexpression. Because limiting **RNR1** expression would be expected to decrease dNTP pools, CLN1 and CLN2 may cause lethality in mec1 strains by causing initiation of DNA replication with inadequate dNTPs. In contrast to mec1 mutants, MEC1 strains with low dNTPs would be able to delay S phase and thereby remain viable. We propose that the essential function for MEC1 may be the same as its checkpoint function during hydroxyurea treatment, namely, to slow S phase when nucleotides are limiting. In a cln1 cln2 background, a prolonged period of expression of genes turned on at the G1-S border, such as **RNR1**, has been observed. Thus deletion of CLN1 and CLN2 could function similarly to overexpression of **RNR1** in suppressing mec1 lethality.

L38 ANSWER 3 OF 29 MEDLINE on STN
ACCESSION NUMBER: 1998448097 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9774971
TITLE: A suppressor of two essential checkpoint genes identifies a novel protein that negatively affects dNTP pools.
AUTHOR: Zhao X; Muller E G; Rothstein R
CORPORATE SOURCE: Department of Genetics and Development, Columbia University, College of Physicians and Surgeons, New York, New York 10032-2704, USA.
CONTRACT NUMBER: GM50237 (NIGMS)
SOURCE: Molecular cell, (1998 Sep) 2 (3) 329-40.
Journal code: 9802571. ISSN: 1097-2765.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 20030304
Entered Medline: 19981102

AB In Saccharomyces **cerevisiae**, MEC1 and RAD53 are essential for cell growth and checkpoint function. Their essential role in growth can be bypassed by deletion of a novel **gene**, SML1, which functions after several genes whose overexpression also suppresses mec1 inviability. In addition, sml1 affects various cellular processes analogous to overproducing the large subunit of ribonucleotide reductase, **RNR1**

. These include effects on mitochondrial biogenesis, on the DNA damage response, and on cell growth. Consistent with these observations, the levels of dNTP pools in sml1 delta strains are increased compared to wild-type. This effect is not due to an increase in RNR transcription. Finally, both in vivo and in vitro experiments show that Sml1 binds to **Rnr1**. We propose that Sml1 inhibits dNTP synthesis posttranslationally by binding directly to **Rnr1** and that Mec1 and Rad53 are required to relieve this inhibition.

L38 ANSWER 4 OF 29 MEDLINE on STN
ACCESSION NUMBER: 97459711 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9315671
TITLE: Rnr4p, a novel ribonucleotide reductase small-subunit protein.
AUTHOR: Wang P J; Chabes A; Casagrande R; Tian X C; Thelander L; Huffaker T C
CORPORATE SOURCE: Section of Biochemistry, Molecular and Cell Biology, Cornell University, Ithaca, New York 14853, USA.
CONTRACT NUMBER: GM40479 (NIGMS)
SOURCE: Molecular and cellular biology, (1997 Oct) 17 (10) 6114-21. Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U30385
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971105
Last Updated on STN: 19971105
Entered Medline: 19971023

AB Ribonucleotide reductases catalyze the formation of deoxyribonucleotides by the reduction of the corresponding ribonucleotides. Eukaryotic ribonucleotide reductases are alpha2beta2 tetramers; each of the larger, alpha subunits possesses binding sites for substrate and allosteric effectors, and each of the smaller, beta subunits contains a binuclear iron complex. The iron complex interacts with a specific tyrosine residue to form a tyrosyl free radical which is essential for activity. Previous work has identified two genes in the yeast Saccharomyces **cerevisiae**, **RNR1** and RNR3, that encode alpha subunits and one **gene**, RNR2, that encodes a beta subunit. Here we report the identification of a second **gene** from this yeast, RNR4, that encodes a protein with significant similarity to the beta-subunit proteins. The phenotype of rnr4 mutants is consistent with that expected for a defect in ribonucleotide reductase; rnr4 mutants are supersensitive to the ribonucleotide reductase inhibitor hydroxyurea and display an S-phase arrest at their restrictive temperature. rnr4 mutant extracts are deficient in ribonucleotide reductase activity, and this deficiency can be remedied by the addition of exogenous Rnr4p. As is the case for the other RNR genes, RNR4 is induced by agents that damage DNA. However, Rnr4p lacks a number of **sequence** elements thought to be essential for iron binding, and mutation of the critical tyrosine residue does not affect Rnr4p function. These results suggest that Rnr4p is catalytically inactive but, nonetheless, does play a role in the ribonucleotide reductase complex.

L38 ANSWER 5 OF 29 MEDLINE on STN
ACCESSION NUMBER: 97459710 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9315670
TITLE: Identification of RNR4, encoding a second essential small subunit of ribonucleotide reductase in Saccharomyces **cerevisiae**.
AUTHOR: Huang M; Elledge S J
CORPORATE SOURCE: Verna and Mars McLean Department of Biochemistry, Howard Hughes Medical Institute, Baylor College of Medicine, Houston, Texas 77030, USA.
CONTRACT NUMBER: GM44664 (NIGMS)
SOURCE: Molecular and cellular biology, (1997 Oct) 17 (10) 6105-13. Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971105
Last Updated on STN: 20020907
Entered Medline: 19971023

AB Ribonucleotide reductase (RNR), which catalyzes the rate-limiting step for deoxyribonucleotide production required for DNA synthesis, is an alpha2beta2 tetramer consisting of two large and two small subunits. RNR2 encodes a small subunit and is essential for mitotic viability in Saccharomyces **cerevisiae**. We have cloned a second essential **gene** encoding a homologous small subunit, RNR4. RNR4 and RNR2 appear to have nonoverlapping functions and cannot substitute for each other even when overproduced. The lethality of RNR4 deletion mutations can be suppressed by overexpression of **RNR1** and RNR3, two genes encoding the large subunit of the RNR enzyme, indicating genetic interactions among the RNR genes. RNR2 and RNR4 may be present in the same reductase complex in vivo, since they coimmunoprecipitate from cell extracts. Like the other RNR genes, RNR4 is inducible by DNA-damaging agents through the same signal transduction pathway involving MEC1, RAD53, and DUN1 kinase genes. Analysis of DNA damage inducibility of RNR2 and RNR4 revealed partial inducibility in dun1 mutants, indicating a DUN1-independent branch of the transcriptional response to DNA damage.

L38 ANSWER 6 OF 29 MEDLINE on STN
ACCESSION NUMBER: 97347514 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9202020
TITLE: Thioredoxin reductase-dependent inhibition of MCB cell cycle box activity in Saccharomyces **cerevisiae**.
AUTHOR: Machado A K; Morgan B A; Merrill G F
CORPORATE SOURCE: Department of Biochemistry and Biophysics and Center for Gene Research and Biotechnology, Oregon State University, Corvallis, Oregon 97331, USA.
SOURCE: Journal of biological chemistry, (1997 Jul 4) 272 (27) 17045-54.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970812
Last Updated on STN: 20020420
Entered Medline: 19970731

AB Mlu1 cell cycle box (MCB) elements are found near the start site of yeast genes expressed at G1/S. Basal promoters dependent on the elements for upstream activating **sequence** activity are inactive in Deltaswi6 yeast. Yeast were screened for mutations that activated MCB reporter genes in the absence of Swi6. The mutations identified a single complementation group. Functional cloning revealed the mutations were alleles of the TRR1 **gene** encoding thioredoxin reductase. Although deletion of TRR1 activated MCB reporter genes, high copy expression did not suppress reporter **gene** activity. The trr1 mutations strongly (20-fold) stimulated MCB- and SCB (Swi4/Swi6 cell cycle box)-containing reporter genes, but also weakly (3-fold) stimulated reporter genes that lacked these elements. The trr1 mutations did not affect the level or periodicity of three endogenous MCB **gene** mRNAs (TMP1, **RNR1**, and SWI4). Deletion of thioredoxin genes TRX1 and TRX2 recapitulated the stimulatory effect of trr1 mutations on MCB reporter **gene** activity. Conditions expected to oxidize thioredoxin (exposure to H2O2) induced MCB **gene** expression, whereas conditions expected to conserve thioredoxin (exposure to hydroxyurea) inhibited MCB **gene** expression. The results suggest that thioredoxin oxidation contributes to MCB element activation and suggest a link between thioredoxin-oxidizing processes such as ribonucleotide reduction and cell cycle-specific **gene** transcription.

L38 ANSWER 7 OF 29 MEDLINE on STN
ACCESSION NUMBER: 96140441 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8552025
TITLE: Overexpression of the **RNR1 gene** rescues Saccharomyces **cerevisiae** mutants in the mitochondrial DNA polymerase-encoding MIP1 **gene**.
AUTHOR: Lecrenier N; Foury F
CORPORATE SOURCE: Unite de Biochimie Physiologique, Universite Catholique de Louvain, Louvain-la-Neuve, Belgium.
SOURCE: Molecular & general genetics : MGG, (1995 Nov 1) 249 (1) 1-7.
Journal code: 0125036. ISSN: 0026-8925.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960306
Last Updated on STN: 19960306
Entered Medline: 19960222

AB A multicopy suppressor **gene** which rescues the temperature-sensitive growth defect of Saccharomyces **cerevisiae** mutants in the mitochondrial DNA (mtDNA) polymerase-encoding MIP1 **gene** has been isolated and identified as the **RNR1 gene**. This **gene**, whose transcript is cell cycle-regulated and mainly expressed at the G1 to S phase transition, encodes the large subunit of ribonucleotide reductase. This enzyme catalyses a limiting step in the production of deoxynucleotides needed for DNA synthesis. The presence of a high copy number of the **RNR1 gene** also decreases the accumulation of rho- mutants observed in diploids that harbour a single copy of the MIP1 **gene**. In cell cycle-synchronised cells, the presence of a high copy number of **RNR1** does not modify its cell cycle transcription regulation and increases its transcript level by a factor of 10 throughout the cell cycle. Our results show that an increased supply of dNTPs in mitochondria can stimulate the mtDNA polymerase activity and indicate that the dNTP concentration may be rate limiting for the replication of mtDNA.

L38 ANSWER 8 OF 29 MEDLINE on STN
ACCESSION NUMBER: 95334366 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7610042
TITLE: The DNA repair genes RAD54 and UNG1 are cell cycle regulated in budding yeast but MCB promoter elements have no essential role in the DNA damage response.
AUTHOR: Johnston L H; Johnson A L
CORPORATE SOURCE: Division of Yeast Genetics, National Institute for Medical Research, London, UK.
SOURCE: Nucleic acids research, (1995 Jun 25) 23 (12) 2147-52.
Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950828
Last Updated on STN: 20020420
Entered Medline: 19950811

AB The DNA repair genes RAD54 and UNG1 have MCB elements in their promoters and are shown to be cell cycle regulated. Their transcripts are coordinately expressed with **RNR1**, ribonucleotide reductase, a MCB-regulated **gene** known to be expressed in late G1. However, no evidence was obtained for a direct role of MCB elements in DNA repair. Of the proteins that bind and activate MCB elements, only mutations in SWI6 have a defect in DNA repair, showing significant sensitivity to methyl methane sulphonate. Furthermore, analysis of the CDC9 promoter indicates that MCB elements are not required for the induction of the **gene** by ultraviolet light irradiation. These promoter elements may not respond directly to DNA damage but may have a role in enhancing the induction response.

L38 ANSWER 9 OF 29 MEDLINE on STN
ACCESSION NUMBER: 95311958 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7791768
TITLE: Disturbance of normal cell cycle progression enhances the establishment of transcriptional silencing in Saccharomyces **cerevisiae**.
AUTHOR: Laman H; Balderes D; Shore D
CORPORATE SOURCE: Department of Microbiology, College of Physicians & Surgeons of Columbia University, New York, New York 10032, USA.
CONTRACT NUMBER: CA09503-0 (NCI)
GM40094 (NIGMS)
SOURCE: Molecular and cellular biology, (1995 Jul) 15 (7) 3608-17. Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199507
ENTRY DATE: Entered STN: 19950807
Last Updated on STN: 20030204
Entered Medline: 19950727

AB Previous studies have indicated that mutation of RAP1 (rap1s) or of the HMR-E silencer ARS consensus element leads to metastable repression of HMR. A number of extragenic suppressor mutations (sds, suppressors of defective silencing) that increase the fraction of repressed cells in rap1s hmr delta A strains have been identified. Here we report the cloning of three SDS genes. SDS11 is identical to SWI6, a transcriptional regulator of genes required for DNA replication and of cyclin genes. SDS12 is identical to **RNR1**, which encodes a subunit of ribonucleotide reductase. SDS15 is identical to CIN8, whose product is required for spindle formation. We propose that mutations in these genes improve the establishment of silencing by interfering with normal cell cycle progression. In support of this idea, we show that exposure to hydroxyurea, which increases the length of S phase, also restores silencing in rap1s hmr delta A strains. Mutations in different cyclin genes (CLN3, CLB5, and CLB2) and two cell cycle transcriptional regulators (SWI4 and MBP1) also suppress the silencing defect at HMR. The effect of these cell cycle regulators is not specific to the rap1s or hmr delta A mutation, since swi6, swi4, and clb5 mutations also suppress mutations in SIR1, another **gene** implicated in the establishment of silencing. Several mutations also improve the efficiency of telomeric silencing in wild-type strains, further demonstrating that disturbance of the cell cycle has a general effect on position effect repression in Saccharomyces **cerevisiae**. We suggest several possible models to explain this phenomenon.

L38 ANSWER 10 OF 29 MEDLINE on STN
ACCESSION NUMBER: 95194366 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1365898
TITLE: DNA synthesis control in yeast: an evolutionarily conserved mechanism for regulating DNA synthesis genes?.
AUTHOR: Merrill G F; Morgan B A; Lowndes N F; Johnston L H
CORPORATE SOURCE: Department of Biochemistry and Biophysics, Oregon State University, Corvallis 97331.
CONTRACT NUMBER: GM24432 (NIGMS)
SOURCE: BioEssays : news and reviews in molecular, cellular and developmental biology, (1992 Dec) 14 (12) 823-30. Ref: 44 Journal code: 8510851. ISSN: 0265-9247.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950425
Last Updated on STN: 19950425

L10 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:86695 BIOSIS
DOCUMENT NUMBER: PREV199800086695
TITLE: Two complementary bioassays for screening the estrogenic potency of xenobiotics: Recombinant **yeast** for trout estrogen receptor and trout hepatocyte cultures.
AUTHOR(S): Petit, F.; Le Goff, P.; Cravedi, J.-P.; Valotaire, Y.; Pakdel, F. [Reprint author]
CORPORATE SOURCE: Equipe Endocrinologie Moleculaire Reproduction, Univ. Rennes I, 35042 Rennes Cedex, France
SOURCE: Journal of Molecular Endocrinology, (Dec., 1997) Vol. 19, No. 3, pp. 321-335. print.
CODEN: JMLEEI. ISSN: 0952-5041.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Feb 1998
Last Updated on STN: 24 Feb 1998

AB A relation between the chemical structure of a xenobiotic and its steroidal action has not yet been clearly established. Thus, it is not possible to define the estrogenic potency of different xenobiotics. An assessment may be accomplished by the use of different bioassays. We have previously developed a **yeast** system highly and stably expressing rainbow trout estrogen receptor (rtER) in order to analyze the biological activity of the receptor. The recombinant **yeast** system appears to be a reliable, rapid and sensitive bioassay for the screening and determination of the direct interaction between ER and estrogenic compounds. This system was used in parallel with a more elaborate biological system, trout hepatocyte aggregate cultures, to examine the estrogenic potency of a wide spectrum of chemicals commonly found in the environment. In hepatocyte cultures, the vitellogenin gene whose expression is principally dependent upon estradiol was used as a biomarker. Moreover, competitive binding assays were performed to determine direct interaction between rtER and xenobiotics. In our study, 50% of the 49 chemical compounds tested exhibited estrogenic activity in the two bioassays: the herbicide diclofop-methyl; the fungicides biphenyl, dodemorph, and triadimefon; the insecticides lindane, methyl parathion, chlordecone, dieldrin, and endosulfan; polychlorinated biphenyl mixtures; the plasticizers or detergents alkylphenols and phthalates; and phytoestrogens. To investigate further biphenyl estrogenic activity, Its principal metabolites were also tested in both bioassays. Among these estrogenic compounds, 70% were able to activate rtER in **yeast** and hepatocytes with variable induction levels according to the system. Nevertheless, 30% of these estrogenic compounds exhibited estrogenic activity in only one of the bioassays, suggesting the implication of metabolites or different pathways in the activation of gene transcription. This paper shows that it is important to combine in vivo bioassays with in vitro approaches to elucidate the mechanism of xenoestrogen actions.

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Entered Medline: 19950411

AB After yeast cells commit to the cell cycle in a process called START, genes required for DNA synthesis are expressed in late G1. Periodicity is mediated by a hexameric **sequence**, known as a MCB element, present in all DNA synthesis **gene** promoters. A complex that specifically binds MCBs has been identified. One polypeptide in the MCB complex is Swi6, a transcription factor that together with Swi4 also binds G1 cyclin promoters and participates in a positive feedback loop at START. The finding that Swi6 is directly involved in both START and DNA synthesis **gene** control suggest a model in which Swi6, activated through its participation in START, serves as the central transcription factor in coordinating late G1 **gene** expression. The mechanism may be conserved in all eukaryotic cells.

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ACCESSION NUMBER: 94141420 EMBASE
DOCUMENT NUMBER: 1994141420
TITLE: The **SAM2 gene** product catalyzes the formation of S-adenosyl-ethionine from ethionine in Saccharomyces **cerevisiae**.
AUTHOR: Martinez-Force E.; Benitez T.
CORPORATE SOURCE: Departamento de Genetica, Facultad de Biologia, Apartado 1095,E-41080 Sevilla, Spain
SOURCE: Current Microbiology, (1994) Vol. 28, No. 6, pp. 339-343. ISSN: 0343-8651 CODEN: CUMIDD
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 940602
Last Updated on STN: 940602

AB Ethionine is the toxic S-ethyl analog of the essential amino acid methionine. Whereas in prokaryotes the ethionine just competes with the methionine, in eukaryotes it can also be transformed into S-adenosyl-ethionine (Ado-Eth), competing with the S-adenosyl-methionine (Ado-Met). When the Ado-Met synthetase activity was studied in strains defective in either of the two isoenzymes, the one coded by the **SAM1 gene** was totally unable to convert ethionine into Ado-Eth and was inhibited by the analog, whereas the enzyme coded by the **SAM2 gene** was able to bind ethionine and was not inhibited by it. This has allowed the development of a procedure to measure Ado-Met synthetase and differentiate between the two isoenzymes present in Saccharomyces **cerevisiae**.

L38 ANSWER 26 OF 29 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 92182190 EMBASE
DOCUMENT NUMBER: 1992182190
TITLE: The Saccharomyces **cerevisiae** GAM2/SIN3 protein plays a role in both activation and repression of transcription.
AUTHOR: Yoshimoto H.; Ohmae M.; Yamashita I.
CORPORATE SOURCE: Center for Gene Science, Hiroshima University,Higashi-Hiroshima 724, Japan
SOURCE: Molecular and General Genetics, (1992) Vol. 233, No. 1-2, pp. 327-330. ISSN: 0026-8925 CODEN: MGGEAE
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 920719
Last Updated on STN: 920719

AB We have cloned GAM2, which is required for transcription of STA1, a **gene** encoding an extracellular glucoamylase in Saccharomyces **cerevisiae** var. diastaticus. DNA **sequence** analysis revealed that GAM2 is the same **gene** as SIN5, known to be a general negative regulator of yeast genes. **RNA** blot analysis indicated that GAM2/SIN3 also acts as a positive regulator of GAM3/ADR6, which in turn is required for transcription of STA1 and ADN2. These results suggest that **SAM2** regulates STA1 expression through transcriptional activation of GAM3 and indicate that GAM2/SIN3 protein is a transcriptional regulator that can play a role in both activation and repression of transcription.

L38 ANSWER 27 OF 29 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:156797 BIOSIS
DOCUMENT NUMBER: PREV199900156797

TITLE: Interaction between the MEC1-dependent DNA synthesis checkpoint and G1 cyclin function in Saccharomyces **cerevisiae.**
AUTHOR(S): Vallen, Elizabeth A. [Reprint author]; Cross, Frederick R.
CORPORATE SOURCE: Dep. Biol., Swarthmore Coll., Swarthmore, PA 19081, USA
SOURCE: Genetics, (Feb., 1999) Vol. 151, No. 2, pp. 458-471. print.
CODEN: GENTAE. ISSN: 0016-6731.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Apr 1999
Last Updated on STN: 16 Apr 1999

AB The completion of DNA synthesis in yeast is monitored by a checkpoint that requires MEC1 and RAD53. Here we show that deletion of the Saccharomyces cereuisiae G1 cyclins CLN1 and CLN2 suppressed the essential requirement for MEC1 function. Wild-type levels of CLN1 and CLN2, or overexpression of CLN1, CLN2, or CLB5, but not CLN3, killed mec1 strains. We identified **RNR1**, which encodes a subunit of ribonucleotide reductase, as a high-copy suppressor of the lethality of mec1 GAL1-CLN1. Northern analysis demonstrated that **RNR1** expression is reduced by CLN1 or CLN2 overexpression. Because limiting **RNR1** expression would be expected to decrease dNTP pools, CLN1 and CLN2 may cause lethality in mec1 strains by causing initiation of DNA replication with inadequate dNTPs. In contrast to mec1 mutants, MEC1 strains with low dNTPs would be able to delay S phase and thereby remain viable. We propose that the essential function for MEC1 may be the same as its checkpoint function during hydroxyurea treatment, namely, to slow S phase when nucleotides are limiting. In a cln1 cln2 background, a prolonged period of expression of genes turned on at the G1-S border, such as **RNR1**, has been observed. Thus deletion of CLN1 and CLN2 could function similarly to overexpression of **RNR1** in suppressing mec1 lethality.

L38 ANSWER 28 OF 29 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1997:65740 BIOSIS
DOCUMENT NUMBER: PREV199799364943
TITLE: Influence of DNA **sequence** identity on efficiency of targeted **gene** replacement.
AUTHOR(S): Negritto, M. Tina; Wu, Xuli; Kuo, Tiffany; Chu, Sheena; Bailis, Adam M. [Reprint author]
CORPORATE SOURCE: Dep. Mol. Biol., Beckman Res. Inst., Div. Gene Therapy, City of Hope National Medical Cent., 1450 E. Duarte Rd., Duarte, CA 91010, USA
SOURCE: Molecular and Cellular Biology, (1997) Vol. 17, No. 1, pp. 278-286.
CODEN: MCEBD4. ISSN: 0270-7306.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 1997
Last Updated on STN: 11 Feb 1997

AB We have developed a system for analyzing recombination between a DNA fragment released in the nucleus from a single-copy plasmid and a genomic target in order to determine the influence of DNA **sequence** mismatches on the frequency of **gene** replacement in Saccharomyces **cerevisiae**. Mismatching was shown to be a potent barrier to efficient **gene** replacement, but its effect was considerably ameliorated by the presence of DNA sequences that are identical to the genomic target at one end of a chimeric DNA fragment. Disruption of the mismatch repair **gene** MSH2 greatly reduces but does not eliminate the barrier to recombination between mismatched DNA fragment and genomic target sequences, indicating that the inhibition of **gene** replacement with mismatched sequences is at least partially under the control of mismatch repair. We also found that mismatched sequences inhibited recombination between a DNA fragment and the genome only when they were close to the edge of the fragment. Together these data indicate that while mismatches can destabilize the relationship between a DNA fragment and a genomic target **sequence**, they will only do so if they are likely to be in the heteroduplex formed between the recombining molecules.

L38 ANSWER 29 OF 29 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1988:481640 BIOSIS
DOCUMENT NUMBER: PREV198886112950; BA86:112950
TITLE: CLONING OF A **GENE** FOR S ADENOSYLMETHIONINE SYNTHESIS IN SACCHAROMYCES-**CEREVISIAE**.
AUTHOR(S): SHIOMI N [Reprint author]; FUKUDA H; MORIKAWA H; FUKUDA Y; KIMURA A
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AB The **gene** for ethionine resistance was isolated, and its phenotypic characteristics were investigated. The cells transformed with this **gene** showed strong resistance to DL-ethionine, and S-adenosylmethionine (SAM) was remarkably accumulated within the cells. Judging from the restriction map of this **gene**, it suggests that the **gene** is not the **gene SAM1** but **SAM2**.

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